

Effects of the transmissibility and virulence of pathogens on intraguild predation in fragmented landscapes



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ABSTRACT

It is well known that pathogenic infection can have a profound effect on the outcome of competition and predation, however the role of pathogenic infection in systems where predators and prey also compete for other resources is yet to be explored (i.e. in systems of intraguild predation). Using a cellular automaton model, we here explore the effect of pathogenic infection on the spatial dynamics of species that also engage in intraguild predation (IGP) in a fragmented landscape. First, the shared pathogen by the predator and prey can enhance species coexistence in the IGP system, consistent with results for non-spatial IGP systems. Second, equilibrium population sizes of the predator and prey depend crucially on the pathogen virulence to the predator but are insensitive to the change in the virulence to the prey. This asymmetric response to virulence change is due to the fact that the predator species has to juggle between predation, resource competition and pathogenic infection. Finally, the response of the pathogen to habitat fragmentation is largely determined by its life-history strategy (transmissibility and virulence) and the trophic level of its host. These results enrich our understanding on the role of pathogens in the ecosystem functioning of eco-epidemiological systems.

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1. Introduction

The interaction between pathogens and their hosts is among the most intimate biotic interactions. Understanding how and to what extent pathogens affect interaction strength, community structure and species distribution is central to theoretical ecology (Anderson and May, 1991; Prenter et al., 2004; Wood, 2006; Hatcher et al., 2006, 2008). To this end, eco-epidemiology as a new branch in mathematical biology focuses especially on integrating epidemiological processes into systems of community ecology (Chattopadhyay and Arino, 1999; Holt and Dobson, 2006; Sieber and Hilker, 2011). Studies in eco-epidemiology have provided increasing insight into understanding the dynamics of complex systems and enhancing the efficacy of conservation management (Chattopadhyay and Arino, 1999; Byers, 2009; Su and Hui, 2011). Current theories have demonstrated that a pathogen can alter or even reverse the outcome of competition, induce complex dynamics in predator–prey systems, affect the structure of species

distribution, and cause trophic cascades in food webs (Lafferty et al., 2006, 2008). Although some eco-epidemiological systems have been examined in depth, knowledge gaps remain, in particular for communities comprising complex biotic interactions. The presence of complex community structures offers a wealth of opportunity for a pathogen to jump from one host species to another, often with unexpected repercussion at the population and community level.

Intraguild predation (IGP) is a common community interaction module and depicts the competition for other resources between predators and their prey (Polis et al., 1989; Arim and Marquet, 2004; Amarasekare, 2006). Although IGP plays an important role in community structure and stability, how pathogens affect the IGP dynamics remains an emerging research focus (Hatcher et al., 2006). The coexistence of predators and prey in an IGP system is possible only if the prey is a superior resource competitor compared to the predator due to the obvious trophic advantage of the predator to its prey (Polis et al., 1989; Holt and Polis, 1997). However, some evidence has shown that pathogens can modify the strength of IGP interaction through both direct and indirect effects. Pathogen-induced change in competitive and foraging abilities can affect the coexistence of multiple predator species in an IGP system (MacNeil et al., 2004; Hatcher et al., 2006; Sieber and Hilker, 2011).

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Specifically, [Hatcher et al., \(2008\)](#) have demonstrated that parasites can broaden the condition of coexistence when the pathogen exerts a greater deleterious effect on the predator.

It is well-known that the success of a transmissible pathogen depends on its life-history traits, including the mode of transmission, the virulence (pathogen-induced mortality of the host), and the immunity of the host ([Morand and Gonzalez, 1997](#); [Hatcher et al., 2008](#); [Horns and Hood, 2012](#); [Webb et al., 2013](#)). In a multi-host system, high virulence and transmissibility can result in the exclusion of the host that is a superior competitor ([Schmitz and Nudds, 1994](#)). An extremely high transmissibility can even lead counter-intuitively, to the disappearance of the pathogen for a long period ([Sun et al., 2010](#)). To this end, it is reasonable to speculate that these life-history traits will also affect the spatial distribution of pathogens and their hosts, especially when pathogenic infection occurs through direct contacts of infected and susceptible individuals ([Rand et al., 1995](#); [Keeling, 1999](#)). The spatial structure in this system could even lead to pathogen-driven extinction ([Webb et al., 2007](#)) and consequently, the spatial structure has been explicitly taken into account when examining the transmission dynamics and the evolution of virulence ([Boots and Sasaki, 1999](#); [Haraguchi and Sasaki, 2000](#); [Boots et al., 2004](#); [Webb et al., 2007, 2013](#); [Su et al., 2008a, 2009a](#)).

An important process that can affect the species distribution and survival is habitat destruction ([Tilman, 1994](#); [Su et al., 2009b](#)). Habitat fragmentation per se is a landscape-level phenomenon in which species that survive in habitat remnants are confronted with a modified environment of reduced area, increased isolation and novel ecological boundaries ([Ewers and Didham, 2006](#); [North and Ovaskainen, 2007](#)). Empirical and theoretical studies have revealed that habitat fragmentation (one component of habitat destruction) can indeed change the behaviour of host–pathogen dynamics ([Ewers and Didham, 2006](#); [Su et al., 2009b](#)). Heterogeneous habitats offer a variety of refuge niches and thus can promote survival and coexistence of species ([Bonsall and Hassell, 2000](#)). As such, fragmented landscapes could become detrimental to the invasion and transmission of pathogens ([Su et al., 2009a](#)). Interestingly, the effect of habitat destruction is also mediated by the life-history traits of the pathogen ([Froeschke et al., 2013](#)). To this end, it is important to examine (1) how the transmission dynamics of pathogens is influenced by the spatial structure of habitat, and (2) how the coupling of habitat structures and pathogen life-history traits affects the dynamics of an IGP system.

Here, we examine the role of habitat destruction in the spatiotemporal dynamics of pathogen transmission in a multi-trophic eco-epidemiological IGP system. Habitat destruction has normally been analysed by making a stipulated fraction of habitat patches unavailable to a focal species ([With, 1997](#)), often in clusters. We develop a cellular automaton (CA) to examine the spatial pattern formation in the IGP system under different levels of habitat destruction and different sets of pathogen life-history traits. CA is the simplest description for nearest neighbour

interactions and is preferred in studies when stochasticity and individual interactions are essential ([Boots and Sasaki, 1999](#); [Haraguchi and Sasaki, 2000](#)).

2. Model

We examined the spatial dynamics of the IGP system in a lattice landscape, with each cell being either suitable or unsuitable to the predators and prey. A fractal landscape was generated using the mid-point displacement algorithm ([With, 1997](#)), and a binary landscape was created by assigning a fraction (h) of cells with the lowest values in the fractal landscape as unsuitable. Both the degree of spatial autocorrelation of the fractal landscape (measured by the roughness constant, H) and the proportion of unsuitable habitat (h) were varied to create an array of complex landscape structures (see [Fig. 1](#) for illustrations). Suitable habitat patches, are thus interspersed among the matrix of unsuitable habitat patches ([Fig. 1](#)).

We considered a transmissive pathogen that affects an IGP system in the above fractal landscape, where the two species in the IGP system (the intraguild predator and prey) compete for resources in the suitable habitat ([Okuyama, 2008](#); [Su et al., 2008b](#)). We, thus, have five possible states of each cell: unsuitable (U), suitable but empty (E), occupied by a susceptible prey (S_N) or an infected prey (I_N), occupied by a susceptible predator (S_P) or an infected predator (I_P). We use the Moore neighbourhood method which considers the eight nearest neighbours engaging in a chess-kings-move in the two-dimensional circular-bounded space. Synchronous updating of all cell states was used in the CA ([Hui and McGeoch, 2007](#); [Su et al., 2009a](#)). The transition rules from step t to $t+1$ are set as follows:

- (i) An empty but suitable cell can be colonized by one susceptible prey or one susceptible predator at a probability of r_n and r_p , respectively. The successful colonizer is randomly chosen from the offspring of neighbouring individuals, with a probability of $1 - (1 - r_n)^{N_{S_N}}$ being a prey and a probability of $1 - (1 - r_p)^{N_{S_P}}$ being a predator ([Rhodes and Anderson, 1997](#)), where N_{S_N} and N_{S_P} are the total number of neighbouring cells with susceptible prey and susceptible predators, respectively.
- (ii) Horizontal transmission describes the movement of a pathogen from one individual to the next through direct or indirect contact, which is consistent with the CA model of local interactions. Thus, pathogenic transmission here only occurs horizontally (through contacts between individuals), not vertically (to progenies), so that the offspring of infected mothers are healthy and susceptible at birth ([Holt and Pickering 1985](#); [Su et al., 2009a](#)). The pathogen can be transmitted from individual i to j through direct contact at a probability of β_{ij} ; for simplicity, we assume $\beta_{ij} = \beta$. Consequently, a susceptible prey can be infected at a probability of

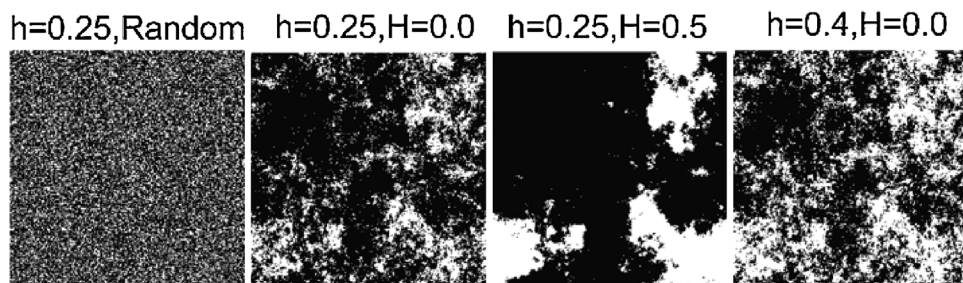


Fig. 1. Three spatial landscape structures in 256×256 lattices, each of which has 25% of habitat loss (white) and a gradient of fragmentation (random is the most fragmented; H refers to the degree of spatial autocorrelation of the fractal landscape patterns).

- $1 - (1 - \beta)^{N_{I_N+I_P}}$, where $N_{I_N+I_P}$ is the sum of neighbouring cells with infected prey and infected predators; similarly, a susceptible predator can be infected at a probability of $1 - (1 - \beta)^{N_{I_N+I_P}}$.
- (iii) Both susceptible and infected predators can consume prey at a probability of λ . A cell with a prey, either susceptible or infected, can be foraged by the neighbouring predators at a probability of $1 - (1 - \lambda)^{N_{S_P+I_P}}$, where $N_{S_P+I_P}$ is the total number of neighbouring predators, susceptible or infected.
- (iv) The natural death rate of prey and predator is m_n and m_p , respectively, and the pathogen-induced death rate (virulence) is μ_n and μ_p , respectively. The death rate of infected prey and predators is, thus, $m_n + \mu_n$ and $m_p + \mu_p$, respectively. The definition of parameters and estimated values are summarized in Table 1.

Here, we considered the scenario of a possible trade-off between the competition ability for basal resources and the predation strength in IGP system; that is, the prey is a superior resource competitor compared to the predator. In particular, to examine the effect of pathogens on the IGP dynamics in a fragmented landscape, we investigated the effect of the transmissibility (β) and the virulence (μ) of the pathogen on the coexistence and spatial structures of the IGP system in a fractal landscape under different levels of habitat destruction (measured by h and H).

3. Results

The spatial patterns of this IGP system under three different levels of pathogen transmissibility were illustrated in a fixed fragmented landscape (Fig. 2). With randomly located initial populations, the IGP system gradually formed a striking spatial wave. Along the spatial wave from the front to the back, the susceptible prey was followed by the infected prey and then by the susceptible and infected predators. Consequently, the susceptible prey was spatially segregated from the infected predators. Such a configuration of the travelling wave could be caused by the following reasons: first, the susceptible hosts might tie each other to reduce the negative impact of pathogen infection; second, the predator–prey and host–pathogen interactions can only occur between adjacent patches. Due to these interactions, pathogens has to encircle (or chase after) susceptible hosts for their possible survival. And also, predators need to chase after the prey for survival. As a consequence susceptible prey is immediately followed by infected prey, which in turn is followed by susceptible and infected predators. Evidently, the travelling wave was also affected by the transmissibility (β) of the pathogen, with a higher transmissibility resulting in a decline of infected predators (Fig. 2).

To further check how the pathogen transmissibility (β) affected the equilibrium population size, we ran the CA model with β

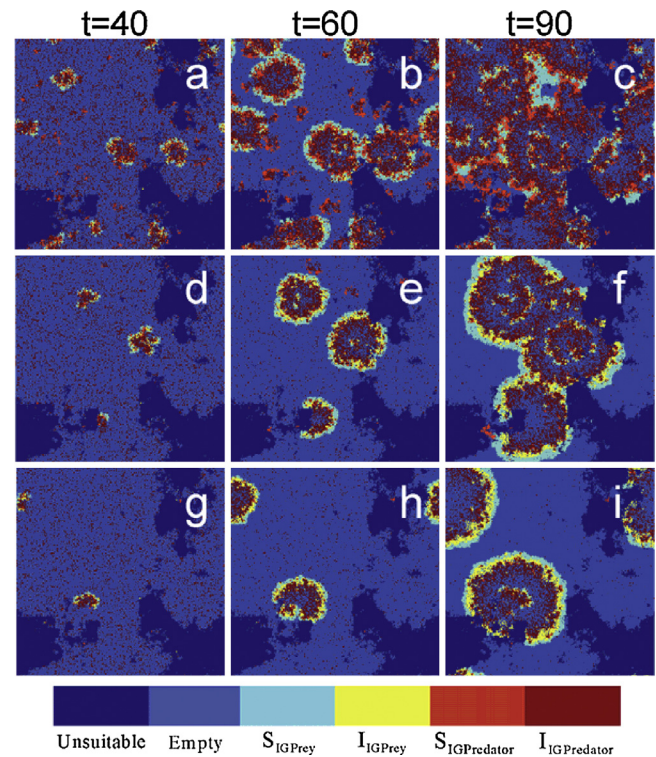


Fig. 2. Effects of transmissibility on the temporal evolution of parasites' spatial patterns under fixed landscape structure. (a–c) $\beta = 0.1$; (d–f) $\beta = 0.2$; (g–i) $\beta = 0.3$. Parameters are $r_n = 0.4$, $r_p = 0.1$, $m_n = 0.05$, $m_p = 0.05$, $\lambda = 0.1$, $\mu_n = 0.005$, $\mu_p = 0.005$ and $h = 0.25$, $H = 0.5$.

ranging from 0 to 0.3 under different levels of fragmentation of habitat (Fig. 3). Here, we choose the simulation parameters to make the IGPrey population extinct under low infection probability, and then the effect of parasite on the coexistence of IGP will be explored dramatically. To weaken the effects of demographic stochasticity, we run five replicate simulations for each given values of infection probability at time $t = 1000$ (the population size is approximate stable in simulation $t = 1000$). The equilibrium population sizes are the average based on the average values from 900 to 1000 time steps in each simulation. First, the prey population size exhibited a nonlinear hump-shape with increasing transmissibility, yet the predator population size declined monotonically under all landscape scenarios. The nonlinear response of the prey to increasing transmissibility arose from a combined interplay of predation, competition and pathogen infection. As the predator is a weaker competitor, the pathogen induced mortality of the predator will always increase with the transmissibility and thus lead to the decline of the predator. Being the stronger competitor, with the increase of transmissibility the

Table 1
Definition of the parameters used in the model.

Parameter	Definition	Default value
h	Proportion of unsuitable habitat	0.25 or 0.4
H	Spatial autocorrelation of fractal landscape	0.0 or 0.5
r_n	Reproduction probability of susceptible prey	0.4
r_p	Reproduction probability of susceptible predator	0.1
β	Transmission probability	–
λ	Predation probability on prey populations	0.1
m_n	Intrinsic mortality rate of prey	0.05
μ_n	Pathogen-induced death rate, then $m_n + \mu_n$ is the enhanced mortality rate due to infection	0.005
m_p	Intrinsic mortality rate of predator	0.05
μ_p	Pathogen-induced death rate, then mortality rate of infected predator is enhanced to $m_p + \mu_p$	–

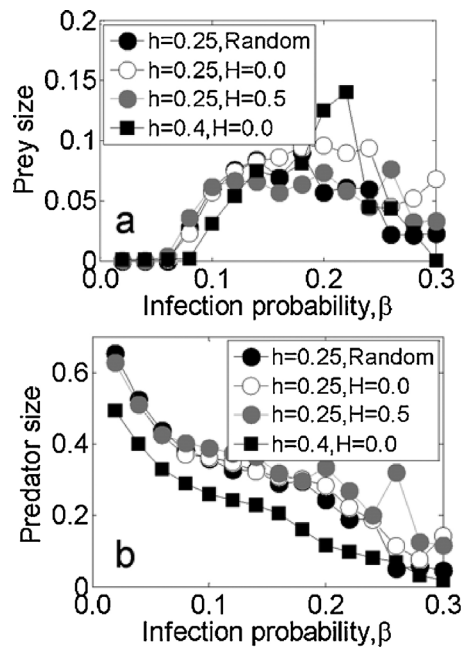


Fig. 3. Effects of transmissibility on population size at equilibrium under three different landscape structures. The equilibrium population sizes are defined as the proportion of lattice occupied by the predator or prey and derived from five replicate simulations for each given values of infection probability at time $t = 1000$. Other parameters are the same with Fig. 2.

prey not only benefits from the decline of predators but is later also detrimentally affected by the spread of pathogens, forming the hump-shaped response. This means that the prey always suffers from the extremes of pathogenic transmissibility, with low population sizes when the transmissibility is either too high or too low.

Habitat fragmentation also affects the spatial dynamics of the IGP system (Fig. 4), forming different travelling waves under different levels of habitat loss and fragmentation. The predator population size suffered from increasing habitat loss (comparing Fig. 4d–f, $h = 0.25$ with Fig. 4j–l, $h = 0.4$), consistent with the results from Fig. 3.

The virulence of the pathogen causes an asymmetric effect on the prey and predators (Fig. 5). Population sizes of the prey and predators were only slightly affected by the increase of pathogen virulence to the prey (μ_n ; Fig. 5a) but responded dramatically to the change of pathogen virulence to the predator (μ_p ; Fig. 5b).

The coupling effect of the virulence μ_p and transmissibility (β) on the equilibrium population size under different levels of habitat structures was further examined (Fig. 6). The prey population size exhibited a hump-shaped response to the change in virulence μ_p for all levels of habitat structures and moderate or high pathogen transmissibility (Fig. 6b and c). The prey population size was ameliorated by low predation when the virulence to the predator was low, and such amelioration disappeared when the virulence became high, which halted the spread of pathogens in the predator population.

The response of the predator to changes in transmissibility and virulence was quite different to that of the prey (Fig. 6d–f). When the transmissibility is low ($\beta = 0.1$), the predator population size increased rapidly with increasing virulence (Fig. 6d). The predator population reached a steady size at high virulence when the prey disappeared. As the transmissibility increases, the response of the predator population size to changing virulence becomes relative slowly increasing (Fig. 6e and f), suggesting a strong effect of pathogen transmissibility on the system. When the virulence is

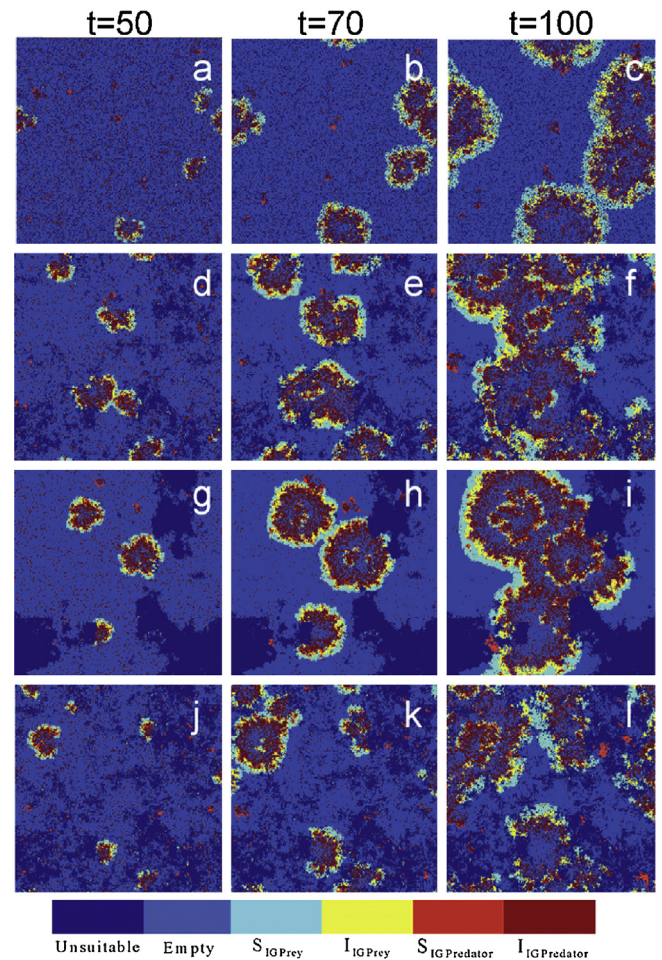


Fig. 4. Temporal evolution of parasites' spatial patterns under various landscape structures. (a–c) $h = 0.25$, random landscape structure; (d–f) $h = 0.25$, $H = 0.0$; (g–i) $h = 0.25$, $H = 0.5$; (j–l) $h = 0.4$, $H = 0.0$. Parameters are $\beta = 0.2$ and others are the same with Fig. 2.

high, the predator increases for all the transmissibility values and habitat structures (Fig. 6e and f), but the prey size decreases except for the highest clumping habitat and transmissibility (Fig. 6a–c). Due to the host–pathogen interaction, the predator increase is attributed to the rapid declines of infected predator by high disease virulence. And the raised size of predator population will reduce the I_{IGPrey} size. Moreover, when the virulence is high, the predator population size reaches its maximum in the random habitat and its minimum in the highly aggregated habitat ($H = 0.5$), in sharp contrast to the scenario when the virulence is low (Fig. 6e and f).

4. Discussion

Pathogens interact with the host differently across trophic levels and thus play a major role in ecosystem functioning (Hatcher et al., 2006, 2008; Wood, 2006; Byers, 2009; Lafferty et al., 2008). Understanding how pathogenic infection affects biotic interactions in wild populations is the challenge in eco-epidemiology (Hatcher et al., 2006; Lafferty et al., 2008). Although many studies have investigated the effect of pathogenic infection on community structures and the outcome of biological invasions (Prenter et al., 2004; Sieber and Hilker, 2011), we here emphasise the spatial dynamics of pathogenic infection in an IGP system under different levels of habitat destruction. Our results showed that a shared pathogen can enhance species coexistence in the IGP system (Fig. 3), confirming the proposition by Hatcher et al., (2008).

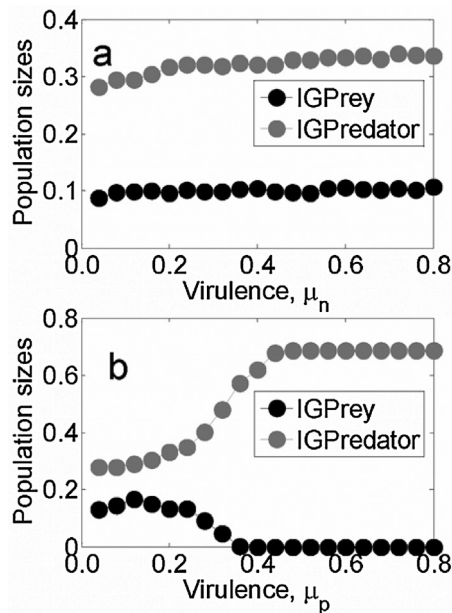


Fig. 5. Long-term (equilibrium) size under virulence with random landscape. (a) The species' size as a function of the virulence in IGPrey (μ_n); (b) the species' size as a function of the virulence in IGPredator (μ_p). Parameters are $\mu_p = 0.005$ for (a) and $\mu_n = 0.005$ for (b). Other parameter values are $r_n = 0.4$, $r_p = 0.1$, $\beta = 0.2$, $\lambda = 0.1$, $m_n = 0.05$, $m_p = 0.05$, $h = 0.25$.

Parasitism increases the range of conditions leading to coexistence in IGP system suggests a combined effects of competition, predation and parasitism (Hatcher et al., 2008, 2014).

Both predators and prey showed drastic responses to the change in virulence to the predator, but were insensitive to the

change in virulence to the prey (Fig. 5). This could be a direct result from the complex interplay of competition, predation and pathogenic infection in the IGP system. The prey has to face the trade-offs between resource acquisition, predator avoidance and pathogenic infection (Borer et al., 2003; Su et al., 2009a), while the predator has to juggle between resource competition and pathogenic infection. To consider the impact of pathogen virulence on community structure and stability, we need to examine the cascade effects across different trophic levels. Other biotic factors, such as dispersal, can also lead to asymmetric responses in the predator and prey (Amarasekare, 2006).

The spreading dynamics of pathogenic infection in response to habitat fragmentation is largely determined by the life history of the pathogen, specifically by its transmissibility, host specificity and virulence (Krasnov and Matthee, 2010; Froeschke et al., 2013). Here, we found that the response of the pathogen to habitat fragmentation was indeed determined by its transmissibility, virulence and the trophic level of its host. Moreover, the ecological impacts of habitat destruction on population dynamics and epidemiological behaviour can be exacerbated by the spatial arrangement of remaining habitat (With, 1997; North and Ovaskainen, 2007). Under the neighbourhood assumption embodied in the CA, habitat fragmentation can reduce habitat quality and accessibility which can intensify interspecific resource competition and reduce the effective range of infection in free-living animal populations (Boots and Sasaki, 1999; Su et al., 2009a). When the virulence is low, aggregated habitats can benefit the inferior competitor (the predator in our case) but be detrimental to the superior competitor (the prey in our case) (Fig. 6). This asymmetric response to habitat fragmentation could be reversed when the virulence is high (Fig. 6). Fragmented habitats may function as a refuge to avoid infection and reduce the infection of susceptible populations. However, high virulence to predators can reverse the refuge effect by the excessive

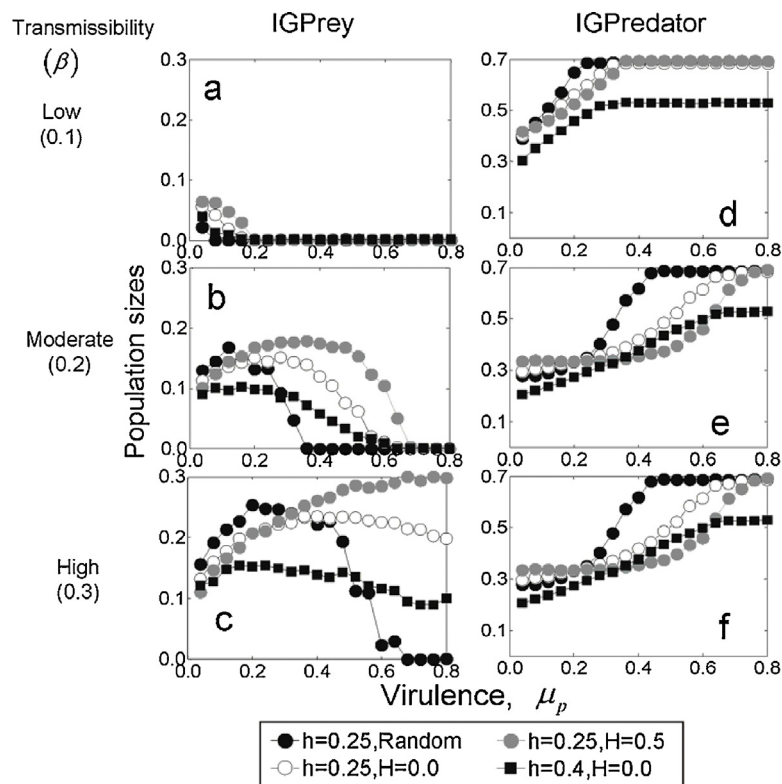


Fig. 6. Long-term (equilibrium) sizes of IGPrey (a–c) and IGPredator (d–f) as a function of virulence in IGPredator (μ_p). The three rows represent increasing levels of transmissibility ($\beta = 0.1, 0.2$ and 0.3). Other parameters are the same with Fig. 5b.

pathogen-induced mortality, consistent with the insular theory (Adler and Levins, 1994).

Results showed that the life history strategy of pathogens and the trophic position of their host determine the effect of habitat heterogeneity on the equilibrium population sizes. This conclusion leads to two future research directions. First, the transmissibility and virulence of a pathogen are important determinants of its invasion and spread. However, the potential trade-off between transmissibility and virulence needs to be considered for predicting the long-term evolution of the pathogen (Boots et al., 2004; Day, 2003). Second, the trade-off between competitive ability and the strength of predation needs to be further investigated. The results reported here only represent one type of trade-off, where the prey is a superior competitor, and the predation strength is strong. Previous studies have showed that variation in competitive ability and the predation strength could also affect species coexistence and the equilibrium population size of intraguild predation systems (Amarasekare, 2006; Hatcher et al., 2008). A general pattern of how intraguild predation communities respond to habitat fragmentation is yet to be developed under different life-history trade-offs.

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